Case Report

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Deceptive Stability: Early Detection of a Contained Rupture of an Infrarenal Abdominal Aortic Aneurysm Presenting as Persistent Abdominal Pain

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ABSTRACT

Background: *Contained rupture of abdominal aortic aneurysm (AAA) represents transient hemodynamic compensation despite ongoing extraluminal blood loss, which may delay diagnosis and definitive intervention. In the absence of hypotension or peritoneal signs, symptoms may be misattributed to benign conditions. This case highlights the importance of early diagnostic escalation and hemodynamic optimization in preventing catastrophic rupture.*

Case Presentation: *A 60-year-old hypertensive female with a significant smoking history presented with persistent infraumbilical pain that progressively worsened over 2 weeks. Initial evaluations, including abdominal radiography and ultrasound, were non-diagnostic except for fecal loading, leading to initial consideration of benign gastrointestinal pathology.*

Upon admission, she remained relatively hemodynamically stable with markedly hypertension (180/120 mmHg) but demonstrated localized infraumbilical tenderness without peritoneal signs. Given persistent pain in a high-risk patient despite an inconclusive initial workup, contrast-enhanced computed tomography (CT) was pursued and revealed a saccular infrarenal aortic abdominal aneurysm. Subsequent aortography demonstrated aneurysmal leakage with perivascular haziness, confirming a contained rupture.

The patient was immediately transferred to the intensive care unit for anti-impulse therapy aimed at reducing aortic wall stress. Blood pressure was controlled to the target systolic range, and heart rate was maintained at 60 to 70 beats per minute through rate control followed by vasodilatory therapy. Emergency surgical repair confirmed posterior wall rupture requiring Dacron graft placement. The intraoperative course was completed on hemodynamic instability requiring vasopressor support and blood transfusion. She was subsequently stabilized and discharged after improvement on intensive care monitoring.

Conclusion: *Contained AAA rupture may present deceptively with stable vital signs and nonspecific abdominal pain; hemodynamic stability should not reassure clinicians when evaluating a high-risk patient with persistent abdominal pain. Early CT imaging, prompt recognition, and aggressive preoperative and postoperative hemodynamic control are critical for preserving the therapeutic window and preventing fatal rupture.*

Keywords

Abdominal aortic aneurysm (AAA), Contained rupture, Infrarenal aortic aneurysm, Aortic rupture, Hemodynamic stability, Anti-impulse therapy.

Introduction

Ruptured abdominal aortic aneurysm (rAAA) is a lethal vascular emergency resulting to classically presents with abdominal, flank, or back pain, hypotension, and a palpable pulsatile abdominal mass; however, this clinical triad is not consistently present at the time of evaluation [1-4]. Up to 5.9% of all rAAA are discovered to be temporarily contained by the surrounding retroperitoneal tissues, allowing relative hemodynamic stability to persist despite the presence of an actual rupture [2,3,5].

This rare presentation causes delayed recognition of rupture, particularly when symptoms are nonspecific and the patient lacks overt signs of hemorrhagic shock or peritoneal irritation. The absence of hypotension should therefore not be considered sufficient evidence against rupture, especially when clinical features raise concern for underlying aortic disease. Contained rupture of abdominal aortic aneurysm poses a diagnostic challenge.

We present a case of a saccular infrarenal AAA with acute contained rupture in a patient who had persistent abdominal pain but remained normotensive and had neither a palpable abdominal mass nor peritoneal signs. The case illustrates how clinical stability may obscure an underlying contained aortic rupture and emphasizes the importance of maintaining suspicion for this diagnosis despite the absence of the classic presentation.

Case Presentation

A 60-year-old woman presented with a chief complaint of persistent infraumbilical abdominal pain. She was a known hypertensive with poor adherence to maintenance medications and had a significant cigarette smoking history of approximately 48 pack-years. She had no known history of diabetes mellitus, coronary artery disease, chronic kidney disease, or previous vascular disease.

Sixteen days prior to admission, the patient developed a brief febrile illness with a temperature of 38–39°C and generalized malaise, which resolved spontaneously the following day. She subsequently developed sudden-onset intermittent infraumbilical abdominal pain, initially rated 6/10, described as burning and crampy and radiating to both flanks. The pain was unrelated to food intake and was associated with early satiety and postprandial, non-projectile vomiting. Nine days prior to admission, persistent abdominal pain prompted emergency consultation; initial complete blood count, urinalysis, and serum creatinine were reportedly unremarkable, and she was treated for presumed acid-peptic disease and musculoskeletal pain with pantoprazole and analgesics. Five days prior to admission, the pain persisted and was accompanied by constipation, prompting a second emergency department consultation. Abdominal radiography showed a normal bowel gas pattern, while ultrasonography demonstrated fecal loading without other significant abnormalities. She was subsequently treated for presumed functional ileus and constipation with bisacodyl and lactulose, while losartan was initiated for newly noted hypertension. Despite symptomatic treatment, the abdominal pain persisted and gradually became more constant. The persistent pain and markedly elevated blood pressure requiring intravenous antihypertensive therapy prompted further medical consultation and hospital admission for evaluation.

On admission, the patient was awake, coherent, ambulatory, and afebrile. Her blood pressure was markedly elevated at 180/120 mmHg, with a heart rate of 110 beats/min, respiratory rate of 23 breaths/min, and oxygen saturation of 98% on room air. Abdominal examination revealed a soft, tympanitic, nondistended abdomen with normoactive bowel sounds and direct tenderness over the infraumbilical region on deep palpation, without guarding, rigidity, abdominal bruit, or a palpable mass. Costovertebral angle tenderness was absent, and peripheral pulses were full and symmetric with preserved capillary refill. Complete blood count revealed leukocytosis with neutrophilic predominance. Other blood examinations were unremarkable.

Despite treatment, the abdominal pain persisted, prompting further diagnostic evaluation. Plain whole-abdominal CT demonstrated segmental calcification of the abdominal aorta and a possible saccular dilation of the distal abdominal aorta immediately proximal to the iliac bifurcation, measuring approximately 2.7 × 3.7 × 5.1 cm. The bowel loops were nondilated, with minimal retained fecal material and no evidence of mechanical obstruction. Subsequent aortography demonstrated a segmentally calcified abdominal aorta with a saccular outpouching arising from the right side of the infrarenal abdominal aorta, approximately 6.6 cm below the renal arteries and above the iliac bifurcation. The aneurysmal sac measured approximately 2.7 × 2.6 cm, with a small communication to the aortic lumen and moderate surrounding perivascular haziness. These findings were interpreted as a saccular AAA with surrounding inflammatory changes, suggestive of a slowly leaking aneurysm with impending rupture. The patient was referred to the Cardiovascular and Cardiothoracic Vascular Surgery services for definitive management. Intravenous nicardipine and esmolol were initiated to maintain a systolic blood pressure of 100–120 mmHg and heart rate of 60–70 beats/min, with adequate analgesia provided. Emergency operative repair was subsequently performed.

Laparotomy revealed a ruptured posterior portion of the infrarenal abdominal aortic aneurysm, confirming a contained rupture. Infrarenal AAA aneurysmorrhaphy with placement of a straight 16-mm Dacron graft was performed. Intraoperatively, the patient developed episodes of hypotension requiring dopamine and norepinephrine for hemodynamic support and received perioperative blood product replacement consisting of 5 units of packed red blood cells, 4 units of fresh frozen plasma, and 4 units of platelet concentrate. Postoperatively, she was transferred to the intensive care unit for close hemodynamic monitoring, with continued nicardipine and esmolol infusions to maintain the target blood pressure and heart rate. Given the antecedent febrile illness, leukocytosis with neutrophilic predominance, elevated procalcitonin, saccular aneurysmal morphology, and perivascular haziness, an infectious etiology was also considered. Infectious Disease consultation was obtained, and empiric antibiotics were initiated. Blood cultures obtained from both upper extremities showed no growth after five days.

By postoperative days 3–4, the patient remained hemodynamically stable, with minimal incisional pain and no wound discharge or erythema. Intravenous antihypertensive therapy was transitioned to oral agents, and ambulation was encouraged. By postoperative days 5–6, she remained comfortable and ambulatory without new complaints. Antibiotic therapy was transitioned to oral ciprofloxacin, and telmisartan was initiated for continued blood pressure control. She was subsequently discharged in stable condition.

Discussion

Up to 50% of patients with an AAA may present with rupture as the initial manifestation [6]. If not promptly diagnosed, rAAA carries an extremely high mortality [4]. Thus, rAAA should remain a diagnostic consideration in patients over 50 years of age presenting with abdominal pain, particularly when accompanied by hemodynamic instability or other features suggestive of vascular disease [4,6].

In the present case, however, the patient had persistent localized infraumbilical pain without hypotension, a palpable abdominal mass, or generalized peritoneal irritation. Instead, she was markedly hypertensive, and her nonspecific symptoms were initially compatible with more common gastrointestinal, musculoskeletal, or genitourinary conditions. This illustrates that the absence of shock does not reliably exclude significant aortic pathology, particularly when rupture remains contained.

Risk factors such as advanced age, cigarette smoking, and hypertension further increase the likelihood of AAA and should heighten clinical suspicion in patients presenting with persistent abdominal pain [7].

AAA usually have a fusiform morphology which typically rupture on larger diameters [2]. On the other hand, saccular morphology is an uncommon form of AAA, comprising approximately 5% of cases, and has been associated with symptomatic presentation and rupture at relatively smaller aneurysm diameters. They have also been described more frequently in women [8,9].

The patient's vascular risk profile should have further increased suspicion for aortic pathology. Established risk factors for AAA include increasing age, male sex, cigarette smoking, low HDL cholesterol, and hypertension [10]. Smoking is among the strongest modifiable risk factors for AAA development and progression, while age, hypertension, and atherosclerotic disease further contribute to risk [11]. In women, smoking appears to be particularly strongly associated with AAA development [9]. Although AAA is more prevalent among men, women have been reported to experience rupture at smaller aneurysm diameters and have higher aneurysm-related mortality [8]. Moreover, a six-year retrospective cohort study found that 33% of patients with rAAA were initially misdiagnosed, with a trend toward more frequent misdiagnosis among women [4]. These findings are particularly relevant to this case, in which an older woman with substantial smoking exposure, poorly controlled hypertension, and evidence of systemic atherosclerosis presented without the classic features of rupture.

The identification of a saccular infrarenal aortic aneurysm in a patient with persistent abdominal pain should raise concern for a symptomatic aneurysm and possible leakage or contained rupture, even when the aneurysm is relatively small and the patient remains hemodynamically stable [9,12]. Although rupture risk in fusiform AAAs generally increases with aneurysm diameter, saccular aneurysms may become symptomatic or rupture at smaller diameters [7]. Saccular AAAs are regarded as a distinct morphological entity involving focal dilation of only part of the aortic circumference. Infection should be excluded and appropriately treated when identified [13]. The optimal management of non-infected saccular AAAs, including the timing of intervention, remains uncertain and requires further research [13]. Nevertheless, their potentially increased rupture risk may justify individualized assessment and consideration of earlier intervention [13].

In the present case, the saccular morphology and associated imaging findings increased concern for leakage. The aneurysm arose focally from the right side of the infrarenal aorta, with a small communication between the aneurysmal sac and aortic lumen and surrounding perivascular haziness. Surgical exploration subsequently demonstrated a posterior wall rupture, confirming that the radiologic abnormality represented a contained rupture rather than an incidental aneurysm.

An infectious or mycotic aneurysm was also considered because of the antecedent febrile illness, leukocytosis with neutrophilic predominance, elevated procalcitonin, saccular morphology, and surrounding perivascular inflammatory changes. A recent febrile illness, particularly in the presence of arterial trauma or instrumentation, bacterial endocarditis, or immunocompromising conditions, should increase suspicion for an infected aneurysm [12]. Imaging features that may suggest infection include saccular or eccentric morphology, multilobulation, perivascular inflammatory changes or fluid, and intramural or perivascular gas [14].

Blood cultures were therefore obtained and empiric antimicrobial therapy was initiated. However, cultures remained negative after five days, and no definite infectious source was identified. Although negative blood cultures do not completely exclude an infected aneurysm, the overall clinical and operative findings favored a degenerative or atherosclerotic aneurysmal process with contained rupture rather than a confirmed infected aneurysm [9].

Imaging was central to establishing the diagnosis. Abdominal duplex ultrasonography is widely used for AAA screening and surveillance because it is rapid, noninvasive, and highly sensitive for detecting aneurysmal dilatation [10]. In this case, however, the patient initially underwent a standard whole-abdominal ultrasonographic examination as part of the evaluation for her abdominal symptoms; this demonstrated fecal loading but did not identify the aortic abnormality. CT was subsequently obtained because of persistent abdominal symptoms and concern for bowel obstruction. CT provides more detailed anatomic characterization and is particularly valuable when symptomatic or ruptured AAA is suspected [12]. Current guidelines recommend CT in hemodynamically stable patients with suspected rAAA to characterize the aneurysm and assess suitability for endovascular repair [9,12]. This case highlights the importance of diagnostic escalation when symptoms persist despite an initially reasonable working diagnosis, particularly in patients with significant vascular risk factors.

Once a symptomatic leaking or ruptured AAA is identified, definitive repair is required. Endovascular repair is generally preferred when appropriate anatomy and institutional expertise permit, while open repair remains an important option when endovascular intervention is unsuitable or unavailable [12]. In this patient, open repair was performed, and operative exploration confirmed the posterior rupture. The affected segment was successfully reconstructed using a straight Dacron graft.

Perioperative hemodynamic management was critical. Before surgery, the patient's marked hypertension warranted control to limit aortic wall stress while preserving adequate organ perfusion. During surgery, however, she developed hypotensive episodes requiring vasopressor support and significant blood-product replacement. This illustrates the narrow hemodynamic balance in ruptured aortic disease. Severe hypertension may increase aortic wall stress and potentially worsen bleeding, whereas excessive reduction in blood pressure or uncontrolled hypotension may compromise vital-organ perfusion [15]. Following repair, the patient underwent close hemodynamic monitoring and subsequently progressed to oral medications, ambulation, and discharge without documented major complications.

This case emphasizes that persistent abdominal pain should not be dismissed as gastrointestinal or musculoskeletal in a patient with significant vascular risk factors simply because hypotension, shock, or a palpable abdominal mass is absent.

Conclusion

Contained rupture of an abdominal aortic aneurysm may present deceptively with stable vital signs and nonspecific abdominal pain, without the classic findings of hypotension, a palpable pulsatile abdominal mass, or peritoneal irritation. In this case, a 60-year-old woman with poorly controlled hypertension and significant cigarette exposure was initially evaluated for more common gastrointestinal causes, with early investigations failing to identify the underlying vascular pathology. Persistence of her symptoms prompted diagnostic escalation, ultimately revealing a saccular infrarenal AAA with radiologic evidence of leakage and operative confirmation of contained rupture.

In summary, this case highlights that hemodynamic stability should not provide false reassurance when a high-risk patient presents with persistent or unexplained abdominal pain. Recognition of vascular risk factors, reassessment when symptoms persist despite initial treatment, early and timely cross-sectional imaging, and prompt vascular surgical evaluation are essential to identifying contained AAA rupture before progression to lethal hemorrhage. Appropriate perioperative hemodynamic control and postoperative monitoring are likewise critical to maintaining this therapeutic window and optimizing outcomes.

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